DISSECTING ANEURYSMS OF AORTA AND LARGE ARTERIES ACCORDING TO PATHOANATOMICAL DATA

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Dissecting aneurysms (DA) of aorta can be established rarely in the clinical and pathoanatomical practice. As a result of all that certain aspects of their etiology, clinical features and morphology are more or less unknown (5, 8, 9, 12, 13).

Recently they are often foundings, but their difficult and rare clinical diagnoses and corresponding therapy (1, 2, 8, 11) make out of such cases a definite problem, which is very actual.

Therefore, the present study is a morphological investigation and clinical as well as morphological analysis of dissecting aneurysms.

Material and methods

The study covers 50 patients died of DA of aorta and large vessels (arteries) for the period of 20 years recently (1962—1981), registered among 13 483 autopsy cases in the Department of pathoanatomy, Higher Institute of Medicine, Varna city. Additional cuts, stained by haematoxilin-eosin (HE), azane after Kruchai, orcein for elastic fibres, tolluidin blue with pH 2 and 4, and RTAN for fibrin. Statistical analysis was done after the method of alternative way.

Results

For the recent 20 years DA of aorta and large arteries were registered in 50 deceased (0.37 %). The frequency of such cases was 8 times increased from 0.08 % (1962—1966) to 0.67 % (1977—1981) with $p<0.01$ (fig. 1).

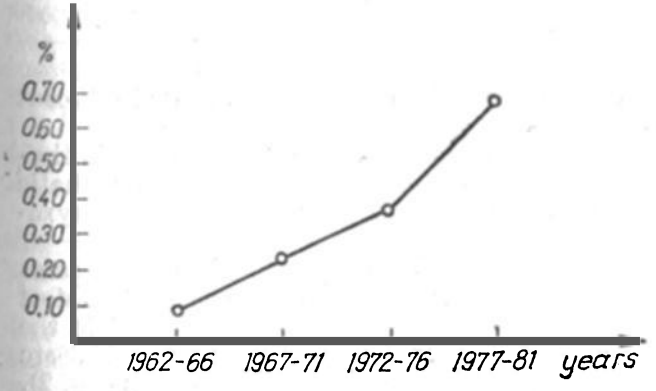

Fig. 1. Frequency of dissecting aneurysms

The ratio between men and women was 2.5:1, but the difference between the frequency of DA in men (0.64±0.21 %) and women (0.40±0.21 %) was not statistically reliable ($p>0.05$).

Bigger number of the studied cases (90 %) were deceased aged over 45 years. There were, however, no statistical differences in the frequency between the various age groups (Table 1).

Table 1

Age groups and frequency of dissecting aneurysms

Age groups	Number of cases	With DA	Percent	Δ	P
26—44	1105	4	0.36	±0.35	—
45—59	2646	16	0.60	±0.29	>0.05
60—74	4052	24	0.59	±0.24	>0,05
75—90	1162	5	0.43	±0.38	>0.05
total	8965	49	0.55	±0.15	—

Etiology factors contributing to a development of DA were very different (Table 2):

Table 2

Analysis of dissecting aneurysms according to etiology

Etiology factor (disease)	Number	Percent	Δ
Arterial hypertension	31	62	±13.45
Atherosclerosis	7	14	± 9.62
Idiopathic medionecrosis	6	12	± 9.00
Marfan's syndrome	3	6	± 6.58
Burning	1	2	± 3.88
Acute myocardial infarction	1	2	± 3.88
Fibromuscular dysplasia	1	2	± 3.88
total	50	100.0	—

The biggest relative part is that of the arterial hypertension. Less significant are the rest etiological factors.

It was established that 7 (14 %) of the deceased had certain disorders in thyroid glands — most often macrofolicular struma and rarer chronical lymphatic thyreoiditis. One of the objects under study had a clinically manifested myxoedema.

In 46 (92 %) deceased DA was of the aorta. Rather rarer — in 4 (8 %) cases were registered separated DA of other arteries: carotic, illiac, encephalic. Most of DA of the aorta shew transverse (cross) or slanting fissures (splittings) in the intima (fig. 2). Most often they were in the upper part (63.04 %) and aortal arc (19.56 %). No fissures in the intima were registered with 4 (8.70 %) cases (fig. 2).

The stratification in the outer third of the media was most often under the influence of mechanical forces (pulse wave) and due to that it propagated (spread-

ed) along the aorta and also the arteries coming out of it. In 43.49 % of all investigated cases with DA the aorta was splitted along its total length. In 28.26 % of the studied deceased limited stratifications were registered in the ascending part of the aorta; they were oftenly combined with an outer rupture of the peri-

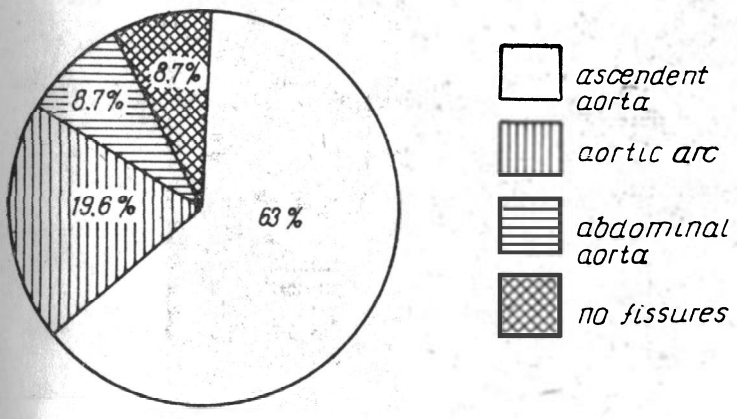

Fig. 2. Localization of intimal fissures in dissecting aneurysms

cardial sack. In 13.04 % were the splittings of ascending part and aortal arc. Rarer (10.87 %) were the separated DA of the abdominal aorta. In 37.78 % of the cases stratification continued along the branches of the aorta. Most often were affected the illiac arteries (15.21 %), mesenterial arteries (6.52 %) and renal arteries (4.35 %).

As it was already mentioned the outer splittings of the aortal wall were most often in the ascending part of the aorta and this was the reason in 63.04 % of the cases to register haemopericarditis. Rarer was established haemothorax (10.87 %). In 19.58 % of the cases there was no rupture of the aneurysms outwards.

The morphological changes in the vessel wall which were most probably the reason for its splitting were quite various in relation to their degree, although they could be very often related to the so called "cyst medionecrosis".

In the biggest group with arterial hypertension the changes in the aortal wall were slightly or moderately expressed and in some of the cases they were even missing. Most often was established accumulation (conglomeration) of metachromatic substance and light looseness, friability and fragmentation of the elastic fibres. In the biggest number of the cases in the adventitia and peripherally of the latter the arterioles were with thicker and hyalinizated walls. In single cases there were formations of arteries with half-close type (fig. 3). In all studied cases the arterioles of the internal organs were with highly expressed hyalinization.

In the group with atherosclerosis the splitting was a result of rupture of vasa vasorum in the projected in the media connective tissue of atherosclerotical plate or a result of stratification of a necrosed atherosclerotical plate (fig. 4).

Most demonstrative were the changes of the vessel wall with cases of idiopathic medionecrosis and Marfan's syndrome. The disorderes in both groups were similar, even identical. Parallelly with the typical foundings of medionecrosis were established large segments with almost totally missing elastic fibres (fig. 5). Besides the fresh dissections very often were registered old ones with already organized masses and projecting connective tissue.

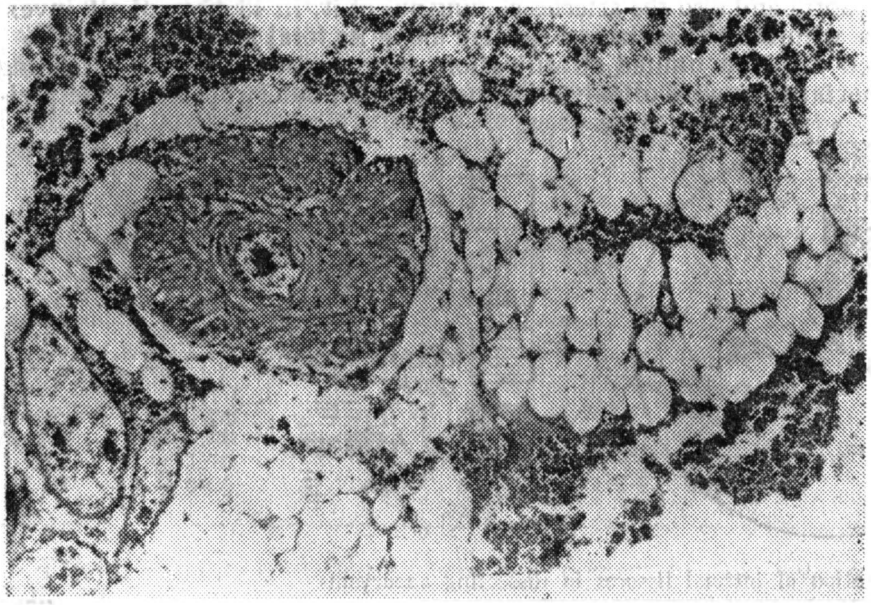

Fig. 3. Periadventitial location of a half-close type artery in hypertension with aortic dissecting aneurysm. Staining HE, magn. 10×6,3

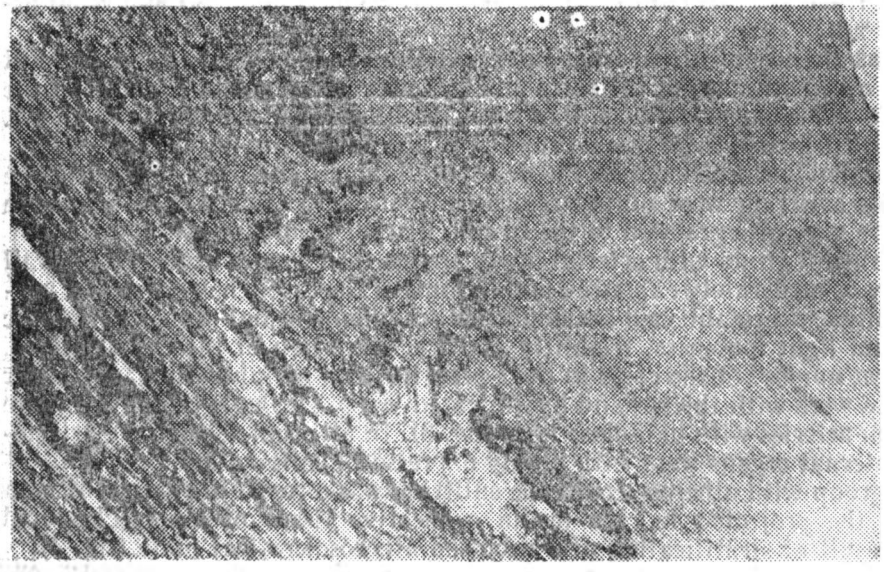

Fig. 4. Macrodissection of aortic wall in the atherosclerotic plaque and necrosis. Staining after Crychay, magn. 10×6,3

Despite of the clear clinical symptoms in 86 % of the investigated cases the diagnosis was clinically proved only in 16 (32 %) objects before death.

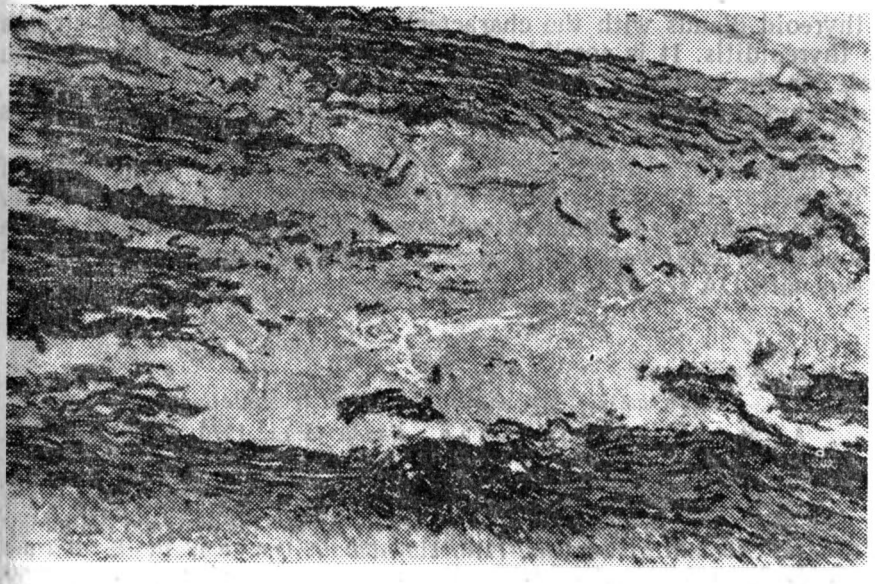

Fig. 5. Severe structural changes of the aortic wall with completely missing elastic fibres in Marfan's syndrome. Staining with orcein for elastic fibres, magn. 10×6,3

The clinical course of such disorders is ususlly acute and heavy. About 2/3 of the patients (64 %) died up to 24th hour, 10 % — until 3rd day, 14 % — 7th day and 12 % — a bit later.

Discussion

The frequency of DA according to the autopsy material varies between 0.30 % and 1.40 % (5, 6, 9). The established in our study frequency is a low one; however, the 8 times increase of the latter for the recent period is quite impressive. This can be explained by the bigger number of DA with arterial hypertension; the same opinion report some other investigators (1, 2, 6, 7, 11, 14). It is obvious that the fluctuation of hypertension, very often contributed to the unproper and regular application of the modern antihypertensive medicines (for example ganglioblockers), plays an important role for the creation of DA (9, 13). The considerable morphological changes in the adventitia and periadventitial vessels which were earlier established by some other authors (3) also have certain significance by the decreased compensative possibilities of the blood flow in the aortal wall.

Special place in our study is contributed to the cases with idiopathic cyst medionecrosis of aorta; no other disorders accompanied the main one. According to F. E. Dalen et al. (7) this is an example of a congenital (innate) deffect of the connective tissue. However, it is quite possible, specially for certain number of the cases, that there are frust forms of Marfan's syndrome (4, 10, 15). The total identity of morphological changes concerning their character and heaviness in cases with Marfan's syndrome and idiopathic medionecrosis confirms the aforementioned suggestion.

It is important that 14 % of the deceased (3 cases with arterial hypertension, 2 with Marfan's syndrome and 1 with idiopathic medionecrosis) had also certain

disorders in thyreoid glands with the characteristics of a macrofollicular struma or lymphatic thyreoiditis. It is possible that the hypothyreoiditis plays also an additional role for the pathogenesis of DA (9, 11).

The propagation of dissections along the length of aorta and its large branches contributes to the variety of clinical diagnosis.

In order to diagnose in a proper way it is obligatory to have in mind most of all DA and to prove this by the modern invasive methods of investigation (8, 10, 14). As for the prognose — the quick and precise operative treatment with a reconstruction of the arterial wall results in very perspective condition for certain number of cases with DA (7, 9, 12).

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РАССЛАИВАЮЩИЕ АНЕВРИЗМЫ АОРТЫ И КРУПНЫХ АРТЕРИЙ, УСТАНОВЛЕННЫХ НА ПАТОЛОГОАНАТОМИЧЕСКИМ ДАННЫМ

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РЕЗЮМЕ

В течение двадцати лет (с 1962 года по 1981 год) на материале 13,403 вскрытий установлено 50 (0,37 %) расслаивающих аневризм аорты и крупных артерий. Отмечается нарастание частоты расслаивающих аневризм приблизительно на 8 раз (от 0,08 % за 2962—1966 г. г. на 0,67 % за 1977—1981 г. г.). 90 % всех исследованных были в возрасте за 45 лет. Установлено, что в 62 % всех случаев развитие аневризм связано с артериальной гипертонией, в 14 % случаев — с атеросклерозом, в 12 % случаев — с идиопатическим медионекрозом и в 6 % случаев — с болезью Марфана. Чаще всего была затронута аорта (92 случая), причем интимальный деффект в 63 % всех случаев обнаруживался в восходящей аорте. Среди умерших с артериальной гипертонией, атеросклерозом, идиопатическим медионекрозом и у больных болезнью Марфана обнаружены существенные различия в морфологических изменениях сосудистой стенки. В первых двух случаях расслоение связано как правило с изменениями стенок сосудов. При остальных двух заболеваниях оно связано с тежелыми изменениями стенки сосуда, сопровождающимися развитием медионекроза.